

Atrioventricular conduction in patients with short PR intervals and normal QRS complexes¹

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His bundle recordings and atrial pacing were used to study 8 patients with short PR intervals and normal QRS complexes. The presence of accelerated conduction above the His bundle was found in all. Six patients had an abnormal response to atrial pacing consisting of an initial period of PH stability. In 3 of these 6 patients the delay in normal PH increase with atrial pacing was partially corrected by propranolol. The data are consistent with a single functional pathway for AV conduction having a variable decrease in the relative refractory period. The consequences of this conduction system include facilitation of rapid repetitive supra-ventricular conduction, and decreased responses to standard doses of propranolol.

In 1952, Lown, Ganong, and Levine described the syndrome of short PR intervals, normal QRS complexes, and paroxysmal supraventricular tachycardia. Though the genesis of the ectopic tachycardia is unclear, the syndrome has been regarded as a type of pre-excitation (Scherf and Cohen, 1964). With the use of the recently described technique of His bundle recording, studies in 3 such patients have revealed short PH and AH times due to accelerated conduction from the low right atrium to the area of the His bundle (Castellanos *et al.*, 1971). The HV interval, representing conduction below the His bundle, was normal in these patients. Conflicting results have reported 3 additional patients in whom the HV time was shortened, suggesting accelerated conduction below the His bundle (Mandel, Danzig, and Hayakawa, 1971). In order to characterize better the atrioventricular conduction system in these patients, electrophysiological studies have been performed in 8 patients with PR intervals less than 120 msec, and the results compared with similar tests in a control group.

Methods

His bundle electrograms were obtained by positioning a bipolar or tripolar catheter in the area of the tricuspid valve (Damato *et al.*, 1969). The PH time was measured from the onset of the P wave in the scalar electrocardiogram to the His bundle potential and the AH time was measured from the onset of the atrial depolarization

complex in the His bundle electrogram. The normal PH time is 80-140 msec and the normal AH interval is 70-120 msec. The HV time was measured from the His potential to the onset of ventricular depolarization in the His electrogram. The normal value is 30-55 msec. The group with short PR intervals consisted of 8 patients with PR intervals less than 120 msec and normal QRS complexes without evidence of Wolff-Parkinson-White conduction. Five patients had a history of documented supraventricular tachycardia and 3 did not. One additional patient with a short PH time was discovered during evaluation for left bundle-branch block and syncope. Three patients (Cases 1, 4, and 9) had coronary artery disease documented by arteriography. The remaining patients had no evidence of heart disease other than arrhythmias. The typical His bundle pattern seen is shown in Fig. 1.

The control group consisted of 10 patients evaluated for atypical chest pain who had normal electrocardiograms and coronary angiograms. There was no history of arrhythmia or conduction disturbance, and the PR interval was greater than 120 msec but less than 200 msec.

The atria were paced from 90 to 150 beats a minute by a gradual reduction in the beat-beat interval, using a Textronic 2600 series pacemaker with pulses 2.5 msec in duration at twice the diastolic threshold. The values are shown for 10-beat increments only. In 5 patients and 10 controls atrial pacing was repeated 10 minutes after the intravenous administration of 0.1 mg/kg propranolol. During atrial pacing the PH interval was measured by the stimulus to His time (SH time as shown in Fig. 3), but for purposes of clarity is referred to as the PH interval in the text. In 4 patients and 10 controls the relative refractory period of the AV conducting system was determined by the extra stimulus method (Wit *et al.*,

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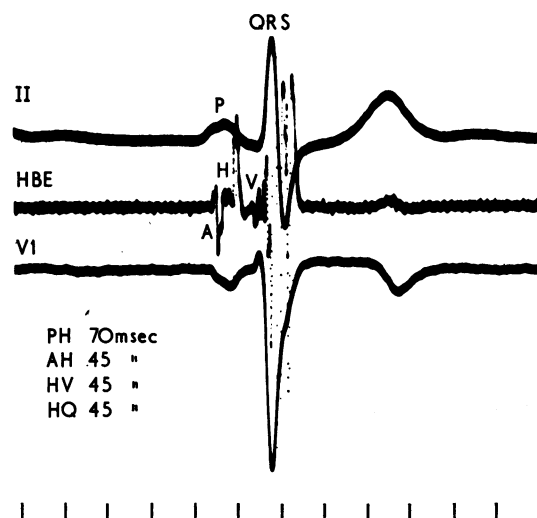


FIG. 1 His bundle electrogram in Case 2 showing a decreased PH and AH interval and a normal HV time. An unusually large His bundle spike is present. II=lead II of the scalar electrocardiogram; V1=lead V1; HBE=His bundle electrogram; A=atrial activation; H=His bundle spike; V=ventricular activation. Time lines are 100 msec.

1970). With the atria paced at rates of 80 to 85 beats a minute premature atrial stimuli were introduced at coupling intervals from 600 to 300 msec at increments of 5-10 msec, and the succeeding pacemaker stimuli were inhibited for 2000 msec by a timed relay circuit to facilitate measurement of the PH and HV intervals. The beginning of the relative refractory period of the AV

conduction system refers to the earliest premature stimulus followed by prolongation of the PH time. In 10 controls the determination of the relative refractory period was repeated after propranolol 0.1 mg/kg. The values given for the relative refractory period in each case refer to the onset of AV conduction delay.

Results

The results of atrial pacing from 90 to 150 beats a minute are shown in the Table. The most striking finding was the presence of a period of stable PH time with atrial pacing in 6 of the 9 patients. The results of atrial pacing in Case 4 are shown in Fig. 2. In contrast, the PH time increased progressively with rate in all of the control patients. These conduction patterns are illustrated in Fig. 3 for 3 patients with accelerated conduction and a representative control.

In comparison with the control group, the patients with PR intervals less than 120 msec had significantly shorter PH times (66 versus 130 msec; $P < 0.01$). The AH interval was less than 60 msec in all patients studied, and greater than 70 msec in all controls. There was no decrease in the HV interval (42 versus 43 msec). The increase in PH time during atrial pacing was less in some of the patients with short PR intervals due primarily to abnormal periods of PH stability as shown in Fig. 3.

After the administration of propranolol to 5 patients the PH interval was seen to increase at lower paced rates as shown in the Table. The response appeared to be proportional to the degree of PH stability with atrial pacing, and was not restored to normal in 3 of the 5 patients. This response was

TABLE Comparison of patients with short PR intervals with controls

Patient	Age (yr)	PR interval (msec)	PH time (msec)	HV time (msec)	Paced rate where PH increase first observed		Total PH increase 90-150 beats min (msec)	Relative refractory period (msec)	Documented tachycardia
Patients (9)					Before propranolol	After propranolol			
Case 1	64	100	60	40	180	140	0	305	None
Case 2	54	115	70	45	150	140	10		Atrial flutter
Case 3	47	*	45	*	140		20	320	None
Case 4	54	85	55	30	140		5	317	Atrial fibrillation
Case 5	46	118	68	50	130	120	45		Atrial fibrillation
Case 6	25	115	65	50	110	90	60		None
Case 7	51	115	75	40	90		70		None
Case 8	53	115	65	40	90	90	30		Atrial fibrillation
Case 9	57	110	70	40	90		30	510	Atrial fibrillation
Average		109 ± 11	66 ± 6	42 ± 6	124 ± 32		30 ± 24		
Controls (10)									
Average		145 ± 13	103 ± 16	43 ± 8	90 ± 0		47 ± 15	638 ± 31	

* This patient had bilateral bundle-branch block (Fig. 2) with a prolonged HV time of 100 msec and was not used in determination of the average values shown.

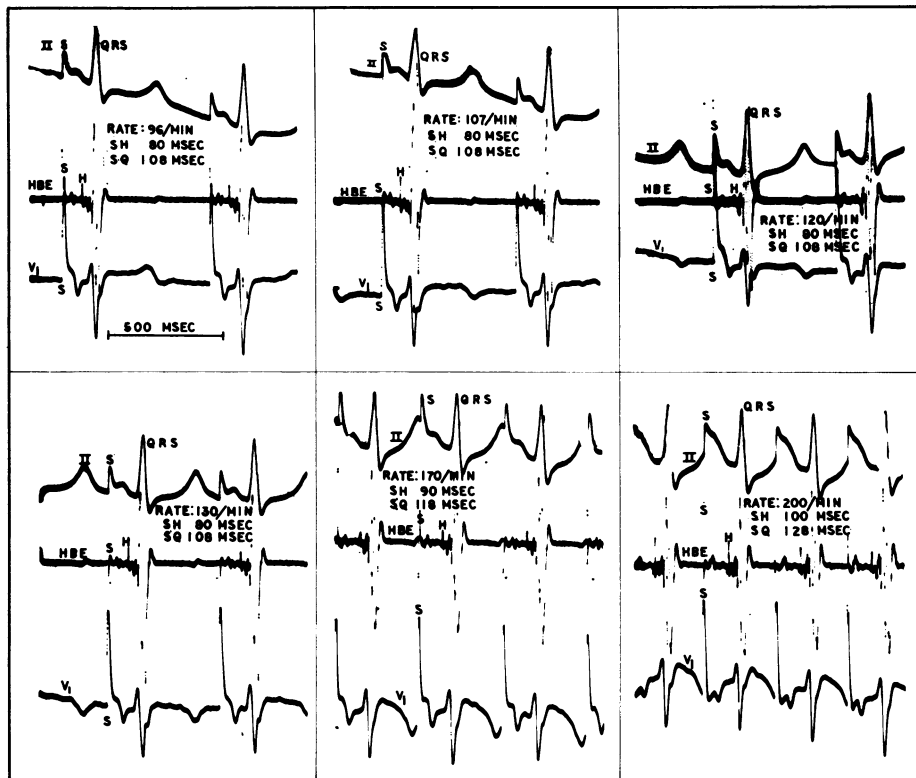


FIG. 2 The effect of atrial pacing atrioventricular conduction in Case 4 is shown by frames extracted from a continuous recording of lead II, V₁, and His bundle electrogram (HBE) as the stimulus (s) to stimulus interval was reduced. The stimulus to His interval (SH) and the stimulus to QRS interval (SQ) remained stable until an atrial rate of 140 beats a minute was reached. At rates greater than 140 beats a minute a gradual increase in the SH and SQ intervals was observed.

different from that seen in the control group, in which a greater PH increase occurred at all paced rates, as previously reported (Smithen, Balcon, and Sowton, 1971).

The onset of the relative refractory period of the AV conduction system was found to be at 305, 317, and 320 msec in 3 patients with conspicuously abnormal PH response to atrial pacing, and at 510 msec in one patient with a normal pacing response. In 10 controls the average relative refractory period began at $638 \text{ msec} \pm 31 \text{ SD}$, and was similar to values previously reported in normal patients (Wit *et al.*, 1970).

Discussion

Studies in normal patients have shown that the PH interval normally increases directly with rate during atrial pacing. This increase has been localized to the area between the AV node and His bundle (Damato

et al., 1969). A similar increase in PH time was seen in all control patients in this series. In contrast, patients with short PR intervals exhibited a spectrum of responses ranging from no increase with atrial pacing until 180 beats a minute to a progressive increase in PH time identical to that of controls. The delay in PH increase with atrial pacing was reflected in the late onset of the relative refractory period measured by the introduction of premature atrial beats in 4 patients. This finding is unique to this form of accelerated AV conduction, and has not been previously reported. This reduction in normal AV nodal delay could permit conduction at a rapid rate similar to that described by Durrer (1968) in a patient with a short PR interval, failure of the PR interval to increase with atrial pacing, and attacks of supraventricular tachycardia at 240/minute. Determination of the rate at which PH prolongation begins during atrial pacing appears to be a suitable

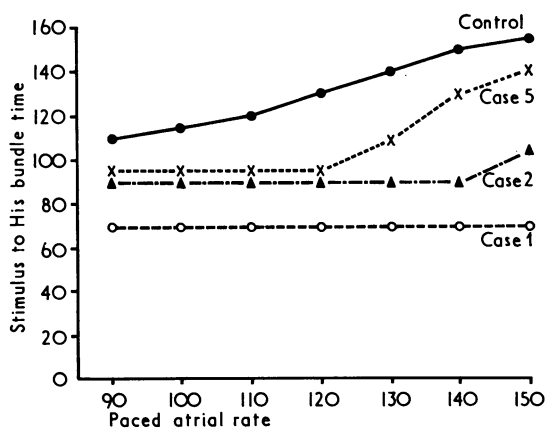


FIG. 3 The effect of atrial pacing on AV conduction is illustrated by plotting the stimulus to His bundle (SH) time versus atrial rate. In the control patients a progressive increase in the SH interval is seen. In 3 patients with short PR intervals a period of PH stability is seen. This period most likely represents a reduction in the onset of the relative refractory period of the AV conducting system.

screening test for reduction in the relative refractory period of the AV conducting system.

Though the precise anatomical basis is unknown, James (1963) has described conduction pathways which appear to bypass a portion of the AV node. These connexions may represent a continuation of the posterior internodal tract, and could avoid part or all of the usual AV conduction delay. An alternate hypothesis is that of specialized fibres within the AV node which have the property of accelerated conduction (Moe, Preston, and Burlington, 1956). It should be noted, however, that no direct evidence of two separate competing pathways was found in these patients. The use of atrial pacing and propranolol produced the expected response, PH prolongation, in both the patients and controls. In the individual patient with a short PR interval the response might not occur until a higher atrial rate was achieved, depending on the presence and degree of the period of PH stability.

Regardless of the exact anatomical connexions, AV conduction in these patients appears to behave as a single functional pathway with a variable decrease in the relative refractory period. In Case 9 with a normal increase in PH time with pacing, the relative refractory period of 510 msec is lower than those recently reported for the AV conduction system in patients with a normal PR interval (Wit *et al.*, 1970). If the effective refractory period of the AV conducting system (the earliest premature response

which does not conduct to the His bundle) was correspondingly reduced, this situation would favour conduction of premature atrial beats with a progressively longer PH time. At very short coupling intervals the PH time could increase conspicuously and initiate supraventricular tachycardia through the re-entry mechanism described by Goldreyer and Damato (1971). Measurement of the effective refractory period was attempted in Case 4 and found to be less than 185 msec. This value is lower than any previously reported in normal patients (Wit *et al.*, 1970). In 12 patients with normal conduction studied in the author's laboratory the average effective refractory period of the AV conducting system was 278 msec \pm 26 SD.

The mechanism responsible for the development of atrial fibrillation and atrial flutter in patients reported in this series is unknown. The initiation of atrial fibrillation by a premature atrial beat in a patient with the Lown-Ganong-Levine syndrome has been reported (Mandel *et al.*, 1971). The onset of atrial fibrillation after a premature atrial beat might be enhanced by the presence of abnormal conduction pathways. The effect of changes in the refractory periods of the AV conducting system on the atrial vulnerable period has not been determined.

Repeat determinations of the relative refractory period in 10 controls after 0.1 mg/kg propranolol have shown the maximal increase to be less than 100 msec. Unless the accelerated AV pathway was unusually sensitive to this drug, one would not expect an initial relative refractory period of 300–350 msec to be restored to normal with this dose. These considerations probably explain the partial response to propranolol seen in Cases 1 and 2.

This pattern of conduction may be encountered in the routine clinical setting since PR intervals of 100 to 120 msec have been described in up to 2 per cent of the normal population (Scherf and Cohen, 1964). The investigator concerned with atrial pacing and the evaluation of drug effects on AV conduction may wish to exclude patients who do not have a normal progressive increase in PH time with increasing rate. The clinician should be aware that these patients may have reduced responses to interventions which normally prolong AV conduction. Patients having conspicuous reductions in the relative refractory period of the AV conduction system may not develop the expected increases in AV conduction time when given propranolol in doses as high as 0.1 mg/kg.

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